



Durable Effects of Concentrated Disadvantage on Verbal Ability among African-American Children

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From the Cover: Inaugural Article

Social Sciences

Durable effects of concentrated disadvantage on verbal ability among African-American children

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ABSTRACT

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Disparities in verbal ability, a major predictor of later life outcomes, have generated widespread debate, but few studies have been able to isolate neighborhood-level causes in a developmentally and ecologically appropriate way. This study presents longitudinal evidence from a large-scale study of >2,000 children ages 6–12 living in Chicago, along with their caretakers, who were followed wherever they moved in the U.S. for up to 7 years. African-American children are exposed in such disproportionate numbers to concentrated disadvantage that white and Latino children cannot be reliably compared, calling into question traditional research strategies assuming common points of overlap in ecological risk. We therefore focus on trajectories of verbal ability among African-American children, extending recently developed counterfactual methods for time-varying causes and outcomes to adjust for a wide range of predictors of selection into and out of neighborhoods. The results indicate that living in a severely disadvantaged neighborhood reduces the later verbal ability of black children on average by ≈ 4 points, a magnitude that rivals missing a year or more of schooling.

Keywords: cognitive ability, neighborhood effects, time-varying causal methods

There is broad consensus among social scientists that cognitive ability predicts adult well-being across a variety of dimensions, whether educational attainment, labor market success, avoidance of criminal behavior, or health. There is also increasing agreement that cognitive ability is significantly shaped early in the life course, such that a focus on children is essential ([1](#)).

Yet strongly divergent positions have been staked out on the sources of cognitive ability and its malleability over childhood. Among the central arguments made in Herrnstein and Murray's widely influential *The Bell Curve* ([2](#)) is the idea that cognitive ability, or what they more generally consider the underlying dimension of intelligence [intelligence quotient (IQ)], is an important explanation for inequality in American society, and that its sources are largely genetic. It follows, they argue, that economic inequality in a knowledge-based meritocratic economy takes on the character of an aristocracy of intelligence not amenable to social policy intervention.

The IQ debate is a longstanding one, but *The Bell Curve* has sparked a resurgence of interest in assessing the malleability of cognitive ability with respect to social environments in school and the home. For example, Winship and Korenman ([3](#)) review existing research and estimate that an additional year of schooling

substantially increases IQ by somewhere between 2 and 4 points. Research on the home environment further suggests that certain parenting styles and cognitive stimulation are positively associated with children's cognitive ability (4).

NEIGHBORHOODS AND COGNITIVE ABILITY.

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Surprisingly little attention has been devoted to the neighborhood as a social context that may influence cognitive ability, despite the fact that children spend a large portion of their daily lives in early childhood engaged in the social life of their neighborhood. We posit that neighborhood residence influences cognitive ability in several ways.

First, observational data suggest that neighborhood poverty is associated with the inconsistency of maternal parenting practices within the home (5, 6), and the strongest findings based on a randomized voucher experiment in the Moving to Opportunity (MTO) program (7) show that moving to neighborhoods with relatively low poverty rates has a substantial positive impact on caregivers' mental health. Hence, there are plausible theoretical reasons to hypothesize that neighborhood disadvantage constrains parental practices and the family environment "under the roof" (8), which may in turn bear on cognitive achievement. Second, because funding of public schools in America is geographically determined, the quality of the school environment is often directly linked to a family's residential location. Third, living in a deeply segregated social and ethnic environment may restrict the speech community to which parents and children are exposed, thus limiting access to academic English. The latter is a potentially key ingredient of success in school and later in the labor market (9, 10) and is measured on tests of verbal ability.

Fourth, and perhaps most important, because of widespread distrust, fear of violence, and isolating physical landscapes (11), severely disadvantaged communities are likely to inhibit speech communication in varied public contexts. A recent body of research has argued for the importance of "communication infrastructures" in the production of civic engagement (12, 13). Independent evidence also suggests that concentrated disadvantage and violence are directly linked to fewer reciprocated exchanges among neighbors outside of the immediate family (14, 15), which implies a restricted range of public verbal interactions and communication infrastructures that children are exposed to as models for learning. The stress of violence in the community in particular may lead parents to isolate themselves out of fear, leading to a restriction in the sorts of social networks and reciprocated exchanges that serve as the building block of social support mechanisms, language development and social skills in verbal encounters. Children's verbal ability and growth potential are thus hypothesized to be diminished by cumulative neighborhood disadvantage.

Unfortunately, very little research sheds direct light on the role of neighborhood disadvantage in explaining verbal ability, and in addition, common strategies for controlling selection preclude unbiased estimation of the magnitude or causal status of neighborhood effects. Many of the covariates in typical regression models represent potential causal pathways by which the neighborhood may influence the outcome. For example, in an attempt to account for characteristics of individuals and families that influence both selection into poor neighborhoods and cognitive outcomes, observational studies often include control variables such as income, family structure, school quality, health problems, employment, and physical disabilities (16, 17). Yet there exists a long line of research positing neighborhood effects on these same factors (18–21). Controlling for endogenous covariates (including school quality) thus has the net result of denying the possibility that there are multiple pathways by which the neighborhood may influence developmental outcomes among children (22). Most observational studies also rest on cross-sectional comparisons where selection bias is difficult to confront. Comparing within-individual changes in cognitive ability associated with stability or change in neighborhood context provides a more direct means to assess the causal effect of social context in observational data.

An alternative approach comes from the MTO experiment, a program that randomly provided housing vouchers to low-income residents of public housing in five cities, allowing them to move into neighborhoods with relatively low poverty (23). Evaluations conducted 3 years after the treatment showed large effects on

various developmental outcomes for some groups, whereas evaluations conducted 4–7 years after treatment showed mostly null effects for the pooled sample across cities as well as for most subgroups, but with a small positive effect on reading scores for African-Americans (7). The MTO treatment effect has been questioned, however, because destination neighborhoods of the experimental group were mostly black (24) and subject to the reinforcing disadvantages of segregation (20).

More relevant to present concerns, MTO families were selected based on poverty, and children grew up in high-poverty neighborhoods. If the effect of neighborhood disadvantage is cumulative, lags, or is most salient early in life, as recent evidence suggests for adolescent mental health (25), moving out of that context in adolescence may not provide the best test of the causal effect of the social environment. Moreover, by design, studies that follow people moving out of poverty do not estimate the impact of moving into poverty. Thus, the MTO experiment, although compelling, does not provide an answer to the central scientific question driving our inquiry.

HYPOTHESIS AND ANALYTIC APPROACH.

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We hypothesize that residing in a severely disadvantaged neighborhood cumulatively impedes the development of academically relevant verbal ability in children. The theoretical notion underlying our work is that spatial disadvantage is encompassed not in a single concurrent characteristic but rather in a synergistic composite of social factors that mark the qualitatively distinct aspect of growing up in truly disadvantaged neighborhoods (18, 20). To consider only neighborhood poverty as the causal treatment of interest is too narrow, because poverty is strongly associated with other ecological characteristics, such as percentage of single-parent families, percentage of family members on welfare and unemployed, and racial segregation (26). We leave for future research to investigate potential mediating mechanisms; the logically prior or first-order task is to assess the causal status of the link between concentrated disadvantage and verbal ability.

Within this framework, we study a representative sample of young people who, in 1995, were growing up in the large and representative urban center of Chicago. Unlike previous researchers, we consider the impact of moving into, as well as out of, disadvantage, wherever that might have occurred in the United States. To do so, we propose a modeling strategy that integrates longitudinal development, in this case sequences of moving across neighborhoods, with new counterfactual methods for time-varying causes and outcomes. Our premise is that research on residential mobility and selection into neighborhood disadvantage are necessary components in assessing the dynamics of neighborhood change and the causal status of neighborhood effects (27, 28). We thus examine the effects of a comprehensive measure of concentrated disadvantage on developmental changes in children's cognitive ability in a sequential cohort study, where selection into neighborhoods is explicitly modeled and, in the first instance, is by adults, not the subjects themselves. The structure of the data allows us to treat neighborhood disadvantage at baseline as a covariate, along with an extensive array of individual and family factors, permitting an explicit multilevel contextual model for selection into later neighborhood treatments that builds on past research assuming *de novo* original contexts or unchanging later neighborhood contexts (e.g., ref. 29). Approximately 17% of black children not living in disadvantage in 1995 moved to a disadvantaged neighborhood some time between 1995 and 2002, and 42% of the population of black children living in disadvantaged neighborhoods in 1995 moved to a nondisadvantaged neighborhood between 1995 and 2002. We exploit these “within-individual” changes to estimate the causal effect of moving.

ESTIMATING CAUSAL EFFECTS.

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We extend the counterfactual account of causality, which conceptualizes causal effects as comparisons among potential outcomes associated with possible treatment assignments (30, 31). In our case, we assume that each person possesses, at time t , a set of potential outcomes corresponding to the sequence of neighborhood treatments that person might have received up to that time. The potential outcome is shaped also by the specific sequence of neighborhoods in which that child has resided up to time t as well as

background characteristics and time-varying covariates. In this scenario, we use marginal structural models (32) and the method of Inverse Probability of Treatment Weighting (IPTW), as adapted by Hong and Raudenbush (33), for the case in which the treatment is enacted collectively on those sharing a social membership at each time. Our data have a structure in which children are cross-classified by neighborhoods over time, and the treatment of interest is at the neighborhood level.

IPTW shares much in common with propensity score stratification or matching (29, 34, 36), where the goal is to create two groups of individuals matched on their propensity to receive the treatment but who differ in the actual treatment received. This strategy has been shown to yield consistent and unbiased estimates of causal effects when treatment assignment is “ignorable,” that is, when there are no unobserved covariates related to the outcome that are also predictive of treatment group assignment once the observed covariates are controlled. The surprising outcome is that, in large samples, matching on the propensity score fully balances the treatment and control groups on all covariates used in modeling the propensity of receiving the treatment, allowing identification of causal effects under the assumption of ignorable treatment assignment. However, propensity score matching is inappropriate for time-varying treatments and outcomes in the presence of time-varying confounding, even if all confounders are observed (32). If later treatments are endogenous to intermediate outcomes of prior treatments, and mediating causal pathways are controlled, both linear adjustments and propensity score matching can produce biased estimates. An example is shown graphically in [supporting information \(SI\) Fig. 2](#).

Robins and colleagues (32, 37) show that such bias can be addressed by fitting a model that weights each subject by the inverse of the predicted probability the subject received the treatment they actually received at a given time point conditional on prior treatment history, time-varying covariate and outcome history, and baseline (time-invariant) covariates. The IPTW approach relies less on information from subjects highly likely to be assigned the treatment status they actually experience. These subjects are “down-weighted.” Subjects who have a low probability of being observed in a given treatment status are “up-weighted,” so we are borrowing more information from them. IPTW thus provides a means to address fundamental problems associated with estimating causal effects of time-varying treatments in the presence of observed time-varying confounding or mediating pathways. IPTW and counterfactual models are no panacea, and they rely fundamentally on having extensive data that measure selection into treatment at each time. Unmeasured covariates that predict treatment assignment even after controlling the observed covariates remain a constant concern. IPTW models, like propensity models, nonetheless have the crucial advantage of forcing investigators to make explicit assumptions about causality, with additional procedures for assessing robustness of results (34).

SPECIFICATION OF MODEL.

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It is first necessary to specify a proper model for selection into treatment status, in our case, living in a neighborhood with concentrated disadvantage. We are aided in this effort by established theory and by prior work on neighborhood attainment, which we draw on to specify a comprehensive model of selection into disadvantaged neighborhoods (28).

Ours is a three-wave study where the first, or baseline, wave is designated as wave 0. Following Hong and Raudenbush (33), we define at wave 1 of data collection two treatments. Let $Z_{1i} = 1$ if the neighborhood in which child i lives at wave 1 is severely disadvantaged; in contrast, $Z_{1i} = 0$ if that neighborhood is not severely disadvantaged. These two possible treatment assignments generate, for each child i at wave 1, potential outcomes $Y_{1i}(1)$, $Y_{1i}(0)$, and causal effect $\Delta_{1i} = Y_{1i}(1) - Y_{1i}(0)$. Similarly, at wave 2, child i is now exposed either to severe neighborhood disadvantage ($Z_{2i} = 1$) or not ($Z_{2i} = 0$), generating now four potential outcomes $Y_{2i}(0,0)$, $Y_{2i}(1,0)$, $Y_{2i}(0,1)$, $Y_{2i}(1,1)$, and the corresponding causal effects:

$$\Delta_{21i} = Y_{2i}(1,0) - Y_{2i}(0,0)$$

(the effect of wave-1 treatment on wave-2 outcome)

$$\Delta_{22i} = Y_{2i}(0,1) - Y_{2i}(0,0)$$

(the effect of wave-2 treatment on wave-2 outcome)

$$\Delta_i^* = Y_{2i}(1,1) - Y_{2i}(0,0) - \Delta_{21i} - \Delta_{22i}$$

(the amplifying effect of 2 consecutive waves

of severe disadvantage on wave-2 outcome). [1]

Under this specification, the wave 2 potential outcome $Y_{2i}(z_1, z_2)$ if child i receives treatment sequence z_1, z_2 is given by

$$Y_{2i}(z_1, z_2) = Y_{2i}(0,0) + z_1\Delta_{21i} + z_2\Delta_{22i} + z_1z_2\Delta_i^* \quad [2]$$

for $z_1, z_2, \in \{0,0;0,1;1,0;1,1\}$. The causal parameters are the corresponding population averages

$$\delta_1 = E(\Delta_{1i}), \delta_{21} = E(\Delta_{21i}), \delta_{22} = E(\Delta_{22i}), \delta^* = E(\Delta_i^*).$$

[3]

The observed outcomes at waves 1 and 2, respectively, are:

$$Y_{1i} = Z_{1i}Y_{2i}(1) + (1 - Z_{1i})Y_{2i}(0)$$

$$Y_{2i} = Z_{1i}Z_{2i}Y_{2i}(1,1) + Z_{1i}(1 - Z_{2i})Y_{2i}(1,0)$$

$$+ (1 - Z_{1i})Z_{2i}Y_{2i}(0,1) + (1 - Z_{1i})(1 - Z_{2i})Y_{2i}(0,0).$$

[4]

The baseline level of neighborhood disadvantage is not used as a causal treatment but is absorbed into a larger vector of pretreatment (time-invariant) covariates X_{0i} for child i . The baseline outcome Y_{0i} is modeled as the first in a time series used to estimate for each child a growth function. We also introduce a vector of time-varying covariates X_{1i} observable at wave 1, after treatment assignment Z_1 but before treatment assignment Z_2 . We can estimate the causal effects of interest (Eq. 3) under the assumption of sequentially strongly ignorable treatment assignment (32). Under this assumption, wave 1 treatment assignment is conditionally independent of the potential outcomes at waves 1 and 2 given the past:

$$Z_1 \perp Y_2(1), Y_2(0) | X_0, Y_0, \quad [5]$$

and wave 2 treatment assignment is conditionally independent of wave 2 potential outcomes given the past:

$$Z_2 \perp Y_2(0,0), Y_2(1,0), Y_2(0,1), Y_2(1,1) | X_0, Y_0, Z_1, X_1, Y_1. \quad [6]$$

We estimate the effects of interest by estimating the cross-classified random effects model:

$$Y_{ti} = \gamma_0 + u_{0i} + \sum_{s=0}^t \sum_{j=1}^J D_{sij} v_j + \varepsilon_{ti} + I(t=1)[\gamma_1 + Z_{1i} \delta_1] + I(t=2)(\gamma_2 + Z_{1i} \delta_{21} + Z_{2i} \delta_{22} + Z_{1i} Z_{2i} \delta^*). \quad [7]$$

[Eq. 7](#) may be regarded as a growth trajectory for each child, except the trajectory is “deflected” by assignments to treatments and neighborhoods. Here, $I(t=1)$ is an indicator taking on a value of unity at time $t=1$ and 0 at other times. Similarly, $I(t=2)$ is an indicator taking on a value of unity at time $t=2$ and 0 at other times. The intercept of this trajectory has a fixed effect γ_0 . The random effect u_{0i} is the child-specific increment to the intercept. The average increase in the outcome between times 0 and 1 for a child who does not experience disadvantage at time 1 is γ_1 . The average increase in the outcome between times 0 and 2 for a child who experiences disadvantage at neither time 1 nor time 2 is γ_2 . The predictor D_{sij} takes on a value of unity if child i lives in neighborhood j at time s . Hence, unlike most previous research even with panel data, we extend Raudenbush and Bryk ([38](#)) to allow neighborhood effects $v_j, j=1, \dots, J$ to cumulate over time. Treatment effects come into the model at appropriate times through the definition of $I(t=1), I(t=2)$. We assume the within-subject random effect is independent and normally distributed, $\varepsilon_{ti} \sim N(0, \sigma^2)$. We make the same assumptions for the neighborhood random effect ($v_j \sim N(0, \Psi^2)$) and the person-specific effects $u_{0i} \sim N(0, \tau^2)$. Given the small sample size relative to a city of nearly 3 million, we also assume no “interference” in treatment between neighborhoods and between subjects within neighborhoods ([39](#)).

Estimation of [Eq. 7](#) would supply unbiased causal inferences if children were assigned randomly to sequences z_1, z_2 of neighborhood disadvantage, but we know this not to be the case ([21](#)). However, if we apply the results of Robins *et al.* ([32](#)) and Hong and Raudenbush ([33](#)) assuming sequentially strongly ignorable treatment assignment ([Eqs. 5](#) and [6](#)), and assuming all of the probabilities defined in [Eq. 8](#) below lie between 0 and 1, we can obtain consistent estimates of the causal effects by applying IPTW. Specifically, for a child receiving treatment sequence z_1, z_2 , we apply weights at waves 0, 1, and 2 as follows (weight construction discussed further below) in estimating [Eq. 7](#):

$$\begin{aligned} w_0 &= 1 \\ w_1(z_1) &= \frac{P(Z_1 = z_1)}{P(Z_1 = z_1 | X_0 = x_0, Y_0 = y_0)} \\ w_2(z_1, z_2) &= w_1(z_1) * \frac{P(Z_2 = z_2 | Z_1 = z_1)}{P(Z_2 = z_2 | Z_1 = z_1, X_0 = x_0, Y_0 = y_0, X_1 = x_1, Y_1 = y_1)}. \end{aligned} \quad [8]$$

DATA.

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The Project on Human Development in Chicago Neighborhoods (PHDCN) offers key analytic advantages for addressing the relationship between verbal ability and neighborhoods. PHDCN longitudinal design begins with an ethnically and socioeconomically diverse population of children ages 0–18 living in a major American city. The 1990 U.S. Census data for Chicago were used to identify 343 neighborhood clusters (NCs), sampling units of one to three census tracts containing $\approx 8,000$ people that were internally similar with respect to distributions of race/ethnicity, socioeconomic status (SES), density, and family structure ([40](#)). A two-stage sampling procedure was used that included selecting a random sample of 80 of 343 Chicago NCs stratified by racial/ethnic composition (seven categories) and SES (high, medium, and low).

Within the final 80 sampled NCs, which include ≈ 200 census tracts, children falling within seven age cohorts

(0, 3, 6, 9, 12, 15, and 18) were sampled from randomly selected households in 1995. This effort led to screening >35,000 households. Dwelling units were selected systematically from a random start within enumerated blocks. Within dwelling units, all households were listed, and age-eligible members were selected with certainty. Participants are representative of children living in a wide range of Chicago neighborhoods (16% European American, 35% African-American, and 43% Latino) and evenly split by gender. Extensive in-home interviews and assessments were conducted with 6,234 sampled children and their primary caregivers at three time points over a 7-year period, at ≈ 2.5 -year intervals (wave 1 in 1995–1997, wave 2 in 1997–1999, and wave 3 in 1999–2002).

Children and their caretakers were followed wherever they moved in the United States. In the present study, we geocoded residential addresses collected at each interview wave and matched them to census tract data for the country as a whole. Almost half the children moved. Followup retention was excellent for an urban sample (75% overall at wave 3). As described below, we explicitly adjust for any selectivity in attrition at each wave.

Because tests of cognitive ability were not given to members of the birth cohort or 3 year olds, our initial analysis is based on the 2,226 children in cohorts 6, 9, and 12 and their caregivers. We exclude subjects in cohorts 15 and 18 because of their unique status as young adults who are making independent choices and decisions about their residential location, and because the measure of verbal ability for the older cohorts is based on a different set of assessments than those used for the younger cohorts. Finally, based on findings elaborated below regarding differential exposure to disadvantage, our main causal inferences pertain to the sample of 772 black children.

MEASURES.

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Verbal cognitive ability. Drawing on previous work using the Chicago data ([41](#), [42](#)), we create a composite measure of subjects' verbal ability based on the results from two widely used tests given to subjects, the Wechsler Intelligence Scale for Children vocabulary test ([43](#)) and the Wide Range Achievement Test reading examination ([44](#)). Overall, the scaled scores from the two tests were correlated at 0.41 at wave 0, 0.48 at wave 1, and 0.54 at wave 2 ($P < 0.001$). These correlations are depressed somewhat by the number of recent immigrants in the overall sample for whom English is not the first or primary language. Among subjects whose parents were born in the U.S., the correlations between the scaled test scores were higher at each time: 0.49 at wave 0, 0.54 at wave 1, and 0.61 at wave 2. For black children, the correlations were also higher at each wave (0.50, 0.55, and 0.58, respectively; $P < 0.001$). To capture the underlying construct with increased measurement precision, we combined the scaled results from each test using principal factor estimation and regression scoring. The first factor, which we retain as our measure of verbal ability, accounts for 71% of the total variance of the factors at wave 0, 75% of the total variance at wave 1, and 74% of the total variance at wave 2. This factor is correlated with each scaled test score at 0.84 at wave 0, 0.86 at wave 1, and 0.86 at wave 2. The resulting scale is normalized to have a mean of 0 and a standard deviation of 15 points.

Concentrated disadvantage. Following and replicating prior work ([40](#)), we focus on six characteristics of census tracts, taken from the 1990 and 2000 U.S. Census, to create a measure of concentrated disadvantage: welfare receipt, poverty, unemployment, female-headed households, racial composition (percentage black), and density of children. We first conducted a principal component analysis and confirmed the emergence of a single factor of concentrated disadvantage, which we then used to generate a regression-weighted scale from the six constituent neighborhood characteristics. Second, we used linear interpolation to impute scale scores in the years between 1990 and 2000 and beyond to match the survey years at each wave.

[Table 1](#) shows factor loadings from the analysis; the first set of results are for loadings using all U.S. census tracts in 1990 and 2000, and the second set shows loadings using only Chicago neighborhoods. The primary difference between the United States as a whole and Chicago neighborhoods (indexed by census tracts) is the prevalence of exposure to concentrated disadvantage among Chicago children. Across all U.S. neighborhoods, the percentage of children <18 years of age exposed to neighborhood disadvantage is far lower than in

Chicago.

Table 1.
Principal component loadings for concentrated disadvantage in 1990 and 2000:
Census tract data for the United States and Chicago

	U.S.		Chicago	
	1990	2000	1990	2000
Factor loadings				
Percentage housing units	0.80	0.87	0.88	0.88
Percentage poverty	0.87	0.87	0.88	0.88
Percentage unemployed	0.88	0.78	0.87	0.88
Percentage female-headed households	0.88	0.88	0.88	0.87
Percentage black children	0.78	0.78	0.88	0.88
Percentage of 0-5 year old	0.87	0.88	0.78	0.78

Table 1.
Principal component loadings for concentrated disadvantage in 1990 and 2000: Census tract data for the United States and Chicago

We define concentrated disadvantage as falling in the bottom quartile of the distribution across Chicago census tracts, the origin of the PHDCN sample. [Table 2](#) shows the frequency of exposure to concentrated disadvantaged by race/ethnicity. The startling result is that no white and only a few Latino families live in the most disadvantaged quartile of Chicago neighborhoods, making it impossible to reliably estimate treatment effects for these groups. This result is not simply attributable to the fact that we include percentage black in our scale of concentrated disadvantage. If we recreate the scale of concentrated disadvantage with no measure of racial composition, the resulting scale is correlated at 0.99 with the initial scale. Race and poverty are ecologically intertwined and thus confounded at the neighborhood level in most large U.S. cities ([20](#)).

Table 2.
African-American, white, and Latino children exposed to alternative neighborhood
disadvantage PHDCN cohorts 6–12

	African-American, White, Latino		
	%	%	%
Treatment definition			
Concentrated disadvantage	Baseline	28	0
High-poverty neighborhoods	Baseline	28	0
Chicago neighborhoods	Baseline	28	0
Concentrated disadvantage	Baseline	28	0
High-poverty neighborhoods	Baseline	28	0
Chicago neighborhoods	Baseline	28	0

Table 2.
African-American, white, and Latino children exposed to alternative neighborhood treatments: PHDCN cohorts 6–12

Nonetheless, we explored alternative definitions of neighborhood disadvantage, as shown in the second and third sets of rows in [Table 2](#). When we define treatment using the bottom quartile of concentrated disadvantage based on the national distribution of neighborhoods, larger numbers of whites and Latinos are exposed to treatment. However, this definition captures virtually all blacks in the sample (e.g., 97% of blacks live in the bottom quartile of U.S. disadvantage at baseline). If we instead define the treatment as living in a neighborhood with >30% poverty, we find exposure among all three race/ethnic groups, although still only 5% of whites are exposed to the treatment under this definition.

The reason for the difference between exposures to treatment using neighborhood poverty vs. the more comprehensive measure of concentrated disadvantage is revealed in the last rows of [Table 2](#). For those exposed to high-poverty neighborhoods at baseline, we see a basic similarity across race in the poverty rate and child density. However, note the profound racial-ethnic differences in other characteristics associated with high-poverty neighborhoods: blacks are much more likely to live in areas of segregation, unemployment, welfare, and concentrated female-headed households. For example, the rate of unemployment is >50% greater in areas where blacks live than those where whites live, and there is a qualitatively different racial composition as well, three-fourths black vs. less than a one-third. Even with the less-stringent poverty treatment, only 18 whites are exposed. Concentrated disadvantage is thus a different treatment than simple poverty and one experienced almost solely by Chicago's black population.

The stratification of America's urban landscape by race precludes the estimation of a single treatment for the entire sample, an important substantive finding and one that has implications for previous efforts comparing racial groups. Indeed, our initial attempts to estimate causal models using pooled race/ethnic samples failed precisely because of the lack of common support in the exposure variable. Only by defining poverty very broadly and ignoring segregation, can we include whites and even most Latinos but at that point virtually all blacks are at risk of exposure to the treatment because of concentrated racial inequality. We therefore focus in this article on the effects of concentrated disadvantage in the lives of black children. By focusing solely on black children, we gain the advantage of eliminating the differences between racial groups in the process of selection into disadvantaged neighborhoods while still being able to study the full distribution of neighborhood environments that blacks experience, recalling again that the sample includes the full range of low-income, middle, and upper-class African-Americans.

[Table 3](#) demonstrates that our definition of concentrated disadvantage induces a wide qualitative difference in

the neighborhood environments of control and treatment groups among blacks. There is nearly a 2:1 difference in exposure to concentrated welfare, unemployment, and poverty between treatment and controls, and there are ~50% differentials for female-headed families and racial segregation. The only minimal difference is for density of children, but this variable has the lowest loading in the scale, and results were not sensitive to its removal.

Characteristic	Treatment (n = 27)	Control (n = 26)
Female	52	50
African American	100	100
Concentrated disadvantage	85	35
Female-headed by neighborhood	55	44
Density of children	55	54
Sample size	27	26

Table 3. Characteristics of treatment neighborhoods compared with control neighborhoods: PHDCN cohorts 6–12, African-American children

BASELINE AND TIME-VARYING COVARIATES.

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Building on prior work analyzing residential moves and neighborhood attainment in the PHDCN data (28), we model selection into concentrated disadvantage with a comprehensive set of time-invariant family- and subject-level covariates along with a set of time-varying covariates. We begin with the age and sex of both subjects and caregivers. Although there was limited variation in immigrant generation among African-Americans, we include a citizenship variable (yes, no) indicating whether the caregiver is a U.S. citizen. The caregiver's educational attainment is measured with four dummy variables indicating whether the caregiver has less than a high school diploma, a high school diploma or a graduate equivalent degree (GED) (the reference group), some college or professional school, or at least a college degree.

We measure several constructs validated elsewhere that tap both the vulnerability and capacity of caretakers in neighborhood selection (28). On the vulnerability side, we include problems with the criminal justice system, violence, and mental health that are known to compromise life-course outcomes. Family criminality represents the number of family members with a criminal record. Domestic violence represents the sum of dichotomous responses to nine survey items asking caregivers about violent or abusive interactions with any current or previous domestic partner. The measure of domestic violence is based on the Revised Conflicts Scale with a 0.84 reliability (45). Caregiver depression is a dichotomous measure coded positively if the caregiver is classified as having experienced a period of major depression in the year before the interview. The measure of major depression is based on the Composite International Diagnostic Interview Short Form.

On the capacity side, social support from community members, including friends and family, has long been considered a means by which parents are able to collectively manage parenting tasks and maintain informal controls over youth. Building on this idea, we conceptualize the social support available to parents as a potentially important influence on the decision to relocate or remain in one's community. The caregiver's perceived level of social support is captured by the mean of 15 survey items on the degree to which the caregiver can rely on friends and family for help or emotional support and the degree of trust and respect between the caregiver and his/her family and friends. The reliability of the scale of social support is 0.77 and predicts a range of social outcomes, including maladaptive parenting.

In addition to the set of stable covariates, we include a set of time-varying covariates that capture change in key aspects of individuals' lives occurring over the course of the survey. The first group relates to employment and economic circumstances and includes the following measures: the employment status of the caregiver and the caregiver's spouse or partner (working or not working); the caregiver's total household income, which consists of six dummy variables indicating whether total household income is below \$10,000, \$10,000–19,999, \$20,000–29,999, \$30,000–39,999 (the reference group), \$40,000–49,999, or \$50,000 and above; a measure of occupational status, which is based on the socioeconomic index (SEI) for caregivers (46); and a dummy variable indicating whether the caregiver is receiving welfare. We also include home ownership, household size (the total number of individuals in the household), and the caregiver's marital status, which consists of dummy variables indicating whether the caregiver is single (the reference group), cohabiting, or married. Descriptive statistics for all covariates are available in [SI Table 6](#).

CONSTRUCTION OF THE WEIGHTS.

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We construct time-varying IPT weights after first modeling selection into disadvantaged neighborhoods. Wave 1 treatment status is predicted by the full set of fixed and time-varying covariates measured at baseline, as well as baseline treatment status and verbal ability. Wave 2 treatment status is predicted by the set of fixed covariates measured at baseline, time-varying covariates measured at baseline and at wave 1, and treatment status and verbal ability measured at both baseline and at wave 1, a total of 43 separate selection predictors. Detailed results for selection into neighborhood disadvantage are available in [SI Table 6](#). Prior treatment is by far the strongest predictor of current treatment, although occupational status, criminality, residence length, social support, employment of caregiver's partner, and marital status independently predict neighborhood disadvantage. In addition, baseline verbal ability, gender, home-owner status, welfare receipt, citizenship, caregiver employment, household size, education, and income are associated at the bivariate level with later neighborhood treatments.

Multivariate selection results form the basis of stabilized inverse probability of treatment weights at waves 1 and 2. As delineated in [Eq. 8](#), the numerator of the wave 1 weight is the unconditional probability of receiving the treatment actually received at wave 1. The denominator of the wave 1 weight is the probability of receiving the treatment actually received, conditional on the full set of baseline covariates described above, including the baseline treatment status (i.e., whether the respondent lived in a disadvantaged neighborhood at baseline) and baseline cognitive ability. The numerator of the wave 2 weight is the probability of receiving the wave 2 treatment actually received, conditional on wave 1 treatment status. The denominator is the probability of receiving the treatment actually received conditional on the full set of covariates measured at waves 1 and 2, prior treatment history, and prior cognitive ability. We multiply the resulting ratio by the wave 1 weight to generate the final wave 2 IPT weights ([Eq. 8](#)). To ensure that the few outlying weights with the largest values do not have a disproportionate impact on our results, we then trimmed the largest weights to the value of the 99th percentile. The weights used in the analysis at time points 1 and 2 are multiplied by a separate weight representing the inverse probability of attrition, thereby also adjusting for selective followup. The final wave 1 and 2 IPT weights, with means of 1.02 (range 0.30 to 11.57) and 0.99 (0.16 to 10.74), respectively, are used in estimating the cross-classified, multilevel [Eq. 7](#) using HLM version 6.30 ([33](#), [38](#)).

Before estimating models of verbal ability, we used a propensity-score stratification approach to ensure that treatment and control group members are balanced on their propensity score as well as key predictors of selection into the treatment ([29](#)). Logit models were used to predict the probability of being exposed to the treatment at wave 1 for African-American children in the final sample. As a conservative test, we then split the sample into 10 equally sized strata based on subjects' propensity scores and examined balance within the strata on the average propensity score, its standard deviation, the predicted logit, exposure to concentrated disadvantage at baseline, and verbal ability at baseline. [Table 4](#) reports the results. Although there are no significant differences within stratum, we focus on patterns and magnitude of differences, because traditional significant tests are not applicable: Balance is a sample rather than population property ([47](#)).

Strata	Treatment	Control	Propensity score	Logit	Verbal ability
1	0.000	0.000	0.000	0.000	0.000
2	0.000	0.000	0.000	0.000	0.000
3	0.000	0.000	0.000	0.000	0.000
4	0.000	0.000	0.000	0.000	0.000
5	0.000	0.000	0.000	0.000	0.000
6	0.000	0.000	0.000	0.000	0.000
7	0.000	0.000	0.000	0.000	0.000
8	0.000	0.000	0.000	0.000	0.000
9	0.000	0.000	0.000	0.000	0.000
10	0.000	0.000	0.000	0.000	0.000

Table 4.

Treatment and control group balance within propensity score strata at wave 1: PHDCN cohorts 6–12, African-American children

Overall, treatment and control group members were extremely well balanced on the propensity score and key covariates in the model. The only strata with any imbalance initially were strata 8 and 10. We thus create a secondary sample that excludes 15 cases from stratum 8 and 47 cases from stratum 10 to generate a sample that is entirely in the area of common support. After excluding these cases, treatment and controls are balanced on their propensity score for treatment at wave 1, as well as on their baseline level of verbal ability. There are differences in baseline verbal ability but mainly in the small-*N* comparisons. More important, the patterns are not consistent or in a direction that favors a treatment effect.

We conducted a similar analysis for treatment status at wave 2 (results not shown). In general, we again found good balance within strata, although there was imbalance on the propensity score in strata 2 and 3. We were able to obtain a fully balanced sample by excluding 77 cases from stratum 2 and 47 cases from stratum 10. Because our models estimate effects at multiple time points, simply eliminating the cases that are outside the area of common support at wave 2 would prohibit us from using these cases to estimate the lagged effects of treatment status at wave 1. We therefore report IPTW results for both the sample that is entirely in the area of common support at wave 1 and the sample entirely in the area of common support at wave 2.

EFFECT ESTIMATES.

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[Table 5](#) displays results from the cross-classified IPTW model assessing the effect of concentrated disadvantage on trajectories of adolescent verbal ability among African-American children. The first set of model parameters reflects the average trajectories of change in verbal ability over the three interview waves. At baseline, African-Americans have scores 1.25 points higher than the overall sample mean (Latinos have scores 3 points below the mean and whites almost 10 points above). Over time, black children's verbal ability grows at a slower rate than the sample as a whole, that is, blacks' scores decline relative to the age-standardized mean in the sample. For example, relative to the sample as a whole, the verbal scores of African-Americans not in the treatment group at waves 1 and 2, respectively, are ≈ 3.5 points lower than at baseline. By the final wave, even African-American children who never experience treatment have scores below the overall sample mean.

	First wave sample of wave 1 (N = 754 children)	First wave sample of wave 2 (N = 754 children)
Intercept	1.25 (0.13)	1.25 (0.13)
Wave 1	-0.48 (0.14)	-0.47 (0.14)
Wave 2	-0.47 (0.14)	-0.47 (0.14)
Effect of wave 1	-0.73 (0.18)	-0.73 (0.18)
Effect of wave 2	-0.73 (0.18)	-0.73 (0.18)
Wave 1 treatment effect	-4.28 (1.78)	-4.28 (1.78)
Wave 2 treatment effect	-3.28 (1.78)	-3.28 (1.78)

[Table 5.](#)

Weighted estimates of the effect of concentrated disadvantage on verbal ability: PHDCN cohorts 6–12, African-American children

The second set of estimates presents best-fitting models for causal effects. The data reveal concentrated disadvantage is not associated with a concurrent deficit in verbal ability. By contrast, wave 1 treatment is estimated to significantly reduce later wave 2 verbal ability by 4.28 points in the balanced sample at wave 1 and 3.28 points in the balanced treatment sample at wave 2 (both $P < 0.05$, two-tailed). Balanced wave 1 treatment is also significant at $P < 0.01$ in a one-tailed test appropriate for the hypothesized direction of treatment effect. In both samples, there is significant variance in the wave 1 causal parameter estimates across neighborhoods, suggesting heterogeneity of treatment effect. The null hypothesis of no treatment effect is thus rejected in two ways: the significance of the effect itself and its variability across neighborhoods.

We estimated an additional set of models assessing the multiplicative effect of living in concentrated disadvantage at waves 1 and 2, net of the main effects at each wave. The results were consistent with the hypothesis that the effects of concentrated disadvantage are interactive, but treatment status at waves 1 and 2 correlates extremely highly with the interaction term representing the sequence of treatments (>0.8). Standard errors were also very large, indicating multicollinearity and lack of precision. We therefore do not report results from this model.

In short, neighborhood effects in our data are not instantaneous but rather are manifested several years later. Based on this finding, we estimated the same wave 1 treatment effect on wave 2 verbal ability but with a completely different method, propensity score matching of individuals ([30](#), [36](#)). We allowed replacement (controls can be matched more than once) and accepted a match only if the difference between the propensity scores of treatment and controls was within 0.01. Using this conservative matching criterion, we obtained a very similar effect estimate among individually matched cases ($n = 144$) to the -4.28 estimate in the balanced sample at wave 1 in [Table 5](#): -4.23 (SE = 2.47, $P < 0.10$ two-tailed; $P < 0.05$, one-tailed). The nonparametric effect estimate averaged across the 10 propensity strata in [Table 4](#) is -3.79 . Because standard errors and hence significance tests in propensity models are considered uncertain ([34](#)), we focus on magnitude and consistency of pattern. Across models, the main result is consistent and robust.

Estimated trajectories of verbal ability associated with living in a severely disadvantaged neighborhood at wave 1 (circa the mid- to late 1990s in Chicago) are displayed graphically in [Fig. 1](#). Scores for black children who lived in such neighborhoods declined sharply relative to the average rate of growth in the sample as a whole, so that by wave 2, their verbal ability scores are well below the mean and ≈ 4 points below those of black children who do not experience the treatment. Although there is variability around the estimate, and it is not possible to extrapolate from these results to make inferences about the long-term trajectories of adults, there is, on average, a large divergence in verbal ability linked to wave 1 neighborhood treatment.

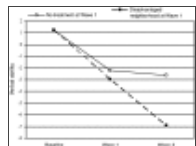


Fig. 1.

Trajectories of verbal ability by neighborhood disadvantage: PHDCN cohorts 6–12, African-American children.

DISCUSSION

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Neighborhoods are not static features of a child's life; instead, neighborhoods change over time as children move through different periods of development, providing unique risks and opportunities at each stage. It follows that neighborhoods have the potential to alter developmental trajectories, and that their influence may be lagged or cumulative. Building on these ideas, we used rich data on selection into and out of neighborhoods to formulate a cross-classified multilevel model designed to estimate causal effects when contextual treatments, outcomes, and confounders all potentially vary over time ([32](#), [33](#), [48](#)). This model was then adapted to generate estimates of the effect of concentrated disadvantage on trajectories of verbal ability.

We estimate that concentrated disadvantage reduces later verbal ability by >4 points, or $>25\%$ of a standard deviation. To put this magnitude in comparison, 1 year in schooling has been associated with between a 2- and 4-point gain in IQ ([3](#)). Thus, we find evidence that the neighborhood environment is an important developmental context for trajectories of verbal cognitive ability. The finding that the strongest effects appear several years after children live in areas of concentrated disadvantage raises important questions about the ways in which neighborhoods may alter growth in verbal ability, producing effects that linger on even if a child leaves a severely disadvantaged neighborhood. Consider trajectories of verbal ability for black children in our study who lived in concentrated disadvantage at wave 1. If one were to randomly provide housing vouchers only to this group and compare outcomes at wave 2, the conclusion would be that there are no neighborhood effects, because there is no difference in verbal ability associated with wave 2 treatment among those receiving treatment in wave 1 ([Table 5](#)). This conclusion would be quite misleading, however, because it brackets the significant lagged effect of living in concentrated disadvantage compared with advantage at wave 1 ([Fig. 1](#)). It follows that residential mobility programs for those who grow up in poverty do not necessarily provide the appropriate test of the causal effect of neighborhood social contexts.

We recognize that our estimates are based on the assumption that unobserved time-invariant and -varying covariates that predict outcomes are unrelated to treatment group assignment after controlling for the observed confounders. There is never a way to definitively test such assumptions, but by limiting our estimates to a balanced sample conditioned on African-American children, we believe our strategy transparently and reasonably addresses longitudinal selection into and out of treatment, an improvement on static estimates. More generally, we believe our proposed method has broad implications for the rapidly growing literature that seeks to estimate causal inferences on longitudinally observed data.

CONCLUSION

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When we consider moves into and out of concentrated disadvantage among a representative sample of black children, not just the poor, durable inequality matters. Indeed, exposure to concentrated disadvantage in Chicago appears to have had detrimental and long-lasting consequences for black children's cognitive ability, rivaling in magnitude the effects of missing 1 year of schooling ([3](#)). Policy discussions of investment in

children are to be applauded ([1](#)), but if our study is any guide, these discussions should be expanded to include a more comprehensive approach to investing in and thereby improving the neighborhood contexts to which children are exposed as they develop cognitive skills crucial for later achievement in life.

SUPPLEMENTARY MATERIAL

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Supporting Information:

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FOOTNOTES

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The authors declare no conflict of interest.

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REFERENCES

[Go to:](#)

1. Heckman JJ. *Science*. 2006;312:1900–1902. [[PubMed](#)]
2. Herrnstein R, Murray C. *The Bell Curve: Intelligence and Class Structure in American Life*. New York: Free; 1994.
3. Winship C, Korenman S. In: *Intelligence, Genes, and Success: Scientists Respond to the Bell Curve*. Devlin B, Fienberg SE, Resnick DP, Roeder K, editors. New York: Springer; 1997. pp. 215–234.
4. Shonkoff JP, Phillips DAE. *From Neurons to Neighborhoods: The Science of Early Childhood Development*, ed National Research Council, Institute of Medicine. Washington, DC: Natl Acad Press; 2000.
5. Klebanov PK, Brooks-Gunn J, McCarton C. *Child Dev*. 1998;69:1420–1436. [[PubMed](#)]
6. Furstenburg FF, Jr, Cook TD, Eccles JS, Elder GH, Jr, Sameroff A. *Managing to Make It: Urban Families and Adolescent Success*. Chicago: Univ of Chicago Press; 1999.
7. Kling J, Liebman J, Katz L. *Econometrica*. 2007;75:83–119.
8. Sampson RJ. In: *Childhood and Violence in the Inner City*. McCord J, editor. New York: Cambridge Univ Press; 1997. pp. 31–77.
9. Ogbu J. *Am Ed Res J*. 1999;36:147–184.
10. Snow CE, Burns SM, Griffin P, editors. *Preventing Reading Difficulties in Young Children: Committee on the Prevention of Reading Difficulties in Young Children*. Washington, DC: Natl Acad Press; 1998.
11. Klinenberg E. *Heat Wave: A Social Autopsy of Disaster in Chicago*. Chicago: Univ of Chicago; 2002.
12. Ball-Rokeach SJ, Kim Y-C, Matei S. *Commun Res*. 2001;28:392–428.
13. Kim Y-C, Ball-Rokeach SJ. *Commun Theor*. 2006;16:173–197.
14. Sampson RJ, Morenoff JD, Earls F. *Am Soc Rev*. 1999;64:633–660.
15. Morenoff JD. *Am J Soc*. 2003;108:976–1017. [[PubMed](#)]
16. Duncan GJ, Boisjoly J, Harris KM. *Demography*. 2001;38:437–447. [[PubMed](#)]
17. Brooks-Gunn J, Duncan G, Kato P, Sealander N. *Am J Soc*. 1993;99:353–395.

18. Wilson WJ. *The Truly Disadvantaged: The Inner City, the Underclass, and Public Policy*. Chicago: Univ of Chicago Press; 1987.
19. Wilson WJ. *When Work Disappears: The World of the New Urban Poor*. New York: Knopf; 1996.
20. Massey DS, Denton N. *American Apartheid: Segregation and the Making of the Underclass*. Cambridge, MA: Harvard Univ Press; 1993.
21. Jencks C, Mayer SE. In: *Inner-City Poverty in the United States*. Lynn LE, McGreary MGH, editors. Washington, DC: Natl Acad Press; 1990. pp. 111–186.
22. Duncan GJ, Raudenbush SW. In: *The Well-Being of Children and Families: Research and Data Needs*. Thornton A, editor. Ann Arbor: Univ of Michigan Press; 2001. pp. 356–383.
23. Goering J, Feins J, editors. *Choosing a Better Life? Evaluating the Moving to Opportunity Social Experiment*. Washington, DC: Urban Institute Press; 2003.
24. Clampet-Lundquist S, Massey DS. *Am J Soc*. 2008 in press.
25. Wheaton B, Clarke P. *Am Soc Rev*. 2003;68:680–706.
26. Massey DS, Eggers ML. *Am J Soc*. 1990;95:1153–1188.
27. Bruch EE, Mare RD. *Am J Soc*. 2006;112:667–709.
28. Sampson RJ, Sharkey P. *Demography*. 2008 in press.
29. Harding D. *Am J Soc*. 2003;109:676–701.
30. Rubin D. *J Ed Psychol*. 1974;66:688–701.
31. Holland P. *J Am Stat Assoc*. 1986;81:945–970.
32. Robins JM, Hernan M, Brumback B. *Epidemiology*. 2000;11:550–560. [[PubMed](#)]
33. Hong G, Raudenbush SW. *J Ed Behav Stat*. 2007 in press.
34. Morgan S, Winship C. *Counterfactuals and Causal Inference: Methods and Principles for Social Research*. New York: Cambridge Univ Press; 2007.
35. Raudenbush S, Bryk A, Cheong FY, Congdon R. *HLM 5: Hierarchical Linear and Nonlinear Modeling*. Chicago: Scientific Software; 2000.
36. Rosenbaum P, Rubin D. *Biometrika*. 1983;70:41–55.
37. Hernán MÁ, Brumback B, Robins JM. *Epidemiology*. 2000;11:561–570. [[PubMed](#)]
38. Raudenbush SW, Bryk AS. *Hierarchical Linear Models: Applications and Data Analysis Methods*. Newbury Park, CA: Sage; 2002.
39. Sobel M. *J Am Stat Assoc*. 2006;101:1398–1407.
40. Sampson RJ, Raudenbush SW, Earls F. *Science*. 1997;277:918–924. [[PubMed](#)]
41. Leventhal T, Xue Y, Brooks-Gunn J. *Child Dev*. 2006;77:1359–1374. [[PubMed](#)]
42. Sampson RJ, Morenoff JD, Raudenbush SW. *Am J Pub Health*. 2005;95:224–232. [[PMC free article](#)] [[PubMed](#)]
43. Wechsler D. *Wechsler Intelligence Scale for Children—Revised*. New York: Psychological Corp; 1974.
44. Wilkinson GC. *WRAT-3: Wide Range Achievement Test, Administration Manual*. Wilmington, DE: Wide Range; 1993.

45. Straus MA, Hamby SL, Boney-McCoy S, Sugarman DB. J Fam Issues. 1996;17:283–316.
46. Nakao K, Treas J. Soc Method. 1994;24:1–72.
47. Imai K, King G, Stuart E. J R Stat Assoc Ser A. 2007 in press.
48. Robins J. Synthese. 1999;121:151–179.

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